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☐ **101:** Mannello F, Gazzanelli G. Related Articles, Links

Tissue inhibitors of metalloproteinases and programmed cell death: conundrums, controversies and potential implications. Apoptosis. 2001 Dec;6(6):479-82. Review. PMID: 11595838 [PubMed - indexed for MEDLINE]

☐ **102:** Benyon RC, Arthur MJ. Related Articles, Links

Extracellular matrix degradation and the role of hepatic stellate cells. Semin Liver Dis. 2001 Aug;21(3):373-84. Review. PMID: 11586466 [PubMed - indexed for MEDLINE]

☐ **103:** Lijnen HR. Related Articles, Links

Plasmin and matrix metalloproteinases in vascular remodeling. Thromb Haemost. 2001 Jul;86(1):324-33. Review. PMID: 11487021 [PubMed - indexed for MEDLINE]

☐ **104:** Creemers EE, Cleutjens JP, Smits JF, Daemen MJ. Related Articles, Links

Matrix metalloproteinase inhibition after myocardial infarction: a new approach to prevent heart failure? Circ Res. 2001 Aug 3;89(3):201-10. Review. PMID: 11485970 [PubMed - indexed for MEDLINE]

☐ **105:** Nguyen M, Arkell J, Jackson CJ. Related Articles, Links

Human endothelial gelatinases and angiogenesis. Int J Biochem Cell Biol. 2001 Oct;33(10):960-70. Review. PMID: 11470230 [PubMed - indexed for MEDLINE]

☐ **106:** Gacko M. Related Articles, Links

[Activation mechanisms, biological role and inhibitors of metalloproteases in the extracellular matrix] Postepy Hig Med Dosw. 2001;55(2):303-18. Review. Polish.

PMID: 11468976 [PubMed - indexed for MEDLINE]

- ❑ 107: [Okazaki I, Watanabe T, Hozawa S, Niioka M, Arai M, Maruyama K.](#) Related Articles, Links



Reversibility of hepatic fibrosis: from the first report of collagenase in the liver to the possibility of gene therapy for recovery.

Keio J Med. 2001 Jun;50(2):58-65. Review.

PMID: 11450593 [PubMed - indexed for MEDLINE]

- ❑ 108: [Opdenakker G, Van den Steen PE, Dubois B, Nelissen I, Van Coillie E, Masure S, Proost P, Van Damme J.](#) Related Articles, Links



Gelatinase B functions as regulator and effector in leukocyte biology.

J Leukoc Biol. 2001 Jun;69(6):851-9. Review.

PMID: 11404367 [PubMed - indexed for MEDLINE]

- ❑ 109: [Licht P, Russu V, Wildt L.](#) Related Articles, Links



On the role of human chorionic gonadotropin (hCG) in the embryo-endometrial microenvironment: implications for differentiation and implantation.

Semin Reprod Med. 2001;19(1):37-47. Review.

PMID: 11394202 [PubMed - indexed for MEDLINE]

- ❑ 110: [Elliott S, Cawston T.](#) Related Articles, Links



The clinical potential of matrix metalloproteinase inhibitors in the rheumatic disorders.

Drugs Aging. 2001;18(2):87-99. Review.

PMID: 11346130 [PubMed - indexed for MEDLINE]

- ❑ 111: [Kossakowska AE, Urbanski SJ, Janowska-Wieczorek A.](#) Related Articles, Links



Matrix metalloproteinases and their tissue inhibitors - expression, role and regulation in human malignant non-Hodgkin's lymphomas.

Leuk Lymphoma. 2000 Nov;39(5-6):485-93. Review.

PMID: 11342332 [PubMed - indexed for MEDLINE]









- ❑ 112: [Curry TE Jr, Osteen KG.](#) Related Articles, Links



Cyclic changes in the matrix metalloproteinase system in the ovary and uterus.

Biol Reprod. 2001 May;64(5):1285-96. Review.

PMID: 11319131 [PubMed - indexed for MEDLINE]

- ☐ **113:** [Kamayama H.](#) Related Articles, Links
 Matrix metalloproteinases and bladder cancer.
J Med Invest. 2001 Feb;48(1-2):31-43. Review.
PMID: 11286015 [PubMed - indexed for MEDLINE]
- ☐ **114:** [Bischof P, Campana A.](#) Related Articles, Links
 A putative role for oncogenes in trophoblast invasion?
Hum Reprod. 2000 Dec;15 Suppl 6:51-8. Review.
PMID: 11261483 [PubMed - indexed for MEDLINE]
- ☐ **115:** [Khasigov PZ, Podobed OV, Ktzoeva SA, Gatagonova TM, Grachev SV, Shishkin SS, Berezov TT.](#) Related Articles, Links
 Matrix metalloproteinases of normal human tissues.
Biochemistry (Mosc). 2001 Feb;66(2):130-40. Review.
PMID: 11255119 [PubMed - indexed for MEDLINE]
- ☐ **116:** [Johnson PR.](#) Related Articles, Links
 Role of human airway smooth muscle in altered extracellular matrix production in asthma.
Clin Exp Pharmacol Physiol. 2001 Mar;28(3):233-6. Review.
PMID: 11236132 [PubMed - indexed for MEDLINE]
- ☐ **117:** [Vincenti MP.](#) Related Articles, Links
 The matrix metalloproteinase (MMP) and tissue inhibitor of metalloproteinase (TIMP) genes. Transcriptional and posttranscriptional regulation, signal transduction and cell-type-specific expression.
Methods Mol Biol. 2001;151:121-48. Review. No abstract available.
PMID: 11217296 [PubMed - indexed for MEDLINE]
- ☐ **118:** [Kraiem Z, Korem S.](#) Related Articles, Links
 Matrix metalloproteinases and the thyroid.
Thyroid. 2000 Dec;10(12):1061-9. Review.
PMID: 11201850 [PubMed - indexed for MEDLINE]
- ☐ **119:** [Stamenkovic I.](#) Related Articles, Links
 Matrix metalloproteinases in tumor invasion and metastasis.
Semin Cancer Biol. 2000 Dec;10(6):415-33. Review.
PMID: 11170864 [PubMed - indexed for MEDLINE]
- ☐ **120:** [Raza SL, Cornelius LA.](#) Related Articles, Links
 Matrix metalloproteinases: pro- and anti-angiogenic activities.
J Investig Dermatol Symp Proc. 2000 Dec;5(1):47-54. Review.

PMID: 11147675 [PubMed - indexed for MEDLINE]

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(CIPO) added

to core patent offices
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research

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L5 ANSWER 30 OF 62 MEDLINE on STN DUPLICATE 24
AN 2002741786 MEDLINE
DN PubMed ID: 12505230
TI Release of matrix metalloproteinases following alcohol septal
ablation in
hypertrophic obstructive cardiomyopathy.
AU Bradham William S; Gunasinghe Himali; Holder Jennifer R; Multani
Marlina;
Killip Donna; Anderson Marianne; Meyer Denise; Spencer William H
3rd;
Torre-Amione Guillermo; Spinale Francis G
CS Medical University of South Carolina, Charleston 29425, USA.
NC HL59165 (NHLBI)
M01 RR01070 (NCRR)
P01 HL48788-08 (NHLBI)
SO Journal of the American College of Cardiology, (2002 Dec 18) 40
(12)
2165-73.
Journal code: 8301365. ISSN: 0735-1097.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200302
ED Entered STN: 20021231
Last Updated on STN: 20030215
Entered Medline: 20030214

L5 ANSWER 31 OF 62 MEDLINE on STN DUPLICATE 25
AN 2002219946 MEDLINE
DN PubMed ID: 11956636
TI Recent advances in the regulation of matrix metalloproteinase 2
activation: from basic research to clinical implication
(Review).
AU Yoshizaki Tomokazu; Sato Hiroshi; Furukawa Mitsuru
CS Department of Otolaryngology, Graduate School of Medicine,
Kanazawa
920-8641, Japan.. tomoy@orl.m.kanazawa-u.ac.jp
SO Oncology reports, (2002 May-Jun) 9 (3) 607-11. Ref: 57
Journal code: 9422756. ISSN: 1021-335X.

CY Greece
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 200210
 ED Entered STN: 20020417
 Last Updated on STN: 20021010
 Entered Medline: 20021008

L5 ANSWER 32 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
 AN 2003:88099 CAPLUS
 DN 138:399731
 TI The inhibitory effect of TIMP-1 on the spontaneous liver fibrosis
 resolution in a transgenic mouse model
 AU Yoshiji, Hitoshi; Yoshii, Junichi; Ikenaka, Yasuhide; Noguchi,
 Ryuichi;
 Yanase, Koji; Namisaki, Tadashi; Fukui, Hiroshi
 CS Third Department of Internal Medicine, Nara Medical University,
 Japan
 SO Japanese Pharmacology & Therapeutics (2002), 30(Suppl. 2),
 S343-S348
 CODEN: JPTABU
 PB Raifu Saiensu Shuppan K.K.
 DT Journal
 LA Japanese

L5 ANSWER 33 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson
 Corporation on
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 AN 2003:155230 BIOSIS
 DN PREV200300155230
 TI Degradation Of Timp-1 And Increased Gelatinase Activity In Human
 Stromal
 Cell Cultures Treated With Nitric Oxide By-products.
 AU Kenney, M. C. [Reprint Author]; Lin, B. [Reprint Author]; Chwa,
 M.
 [Reprint Author]; Saghizadeh, M. [Reprint Author]; Rosenberg, S.
 [Reprint
 Author]; Ljubimov, A. V. [Reprint Author]; Brown, D. J. [Reprint
 Author]
 CS Ophthalmology Research, Cedars-Sinai Medical Center, Los
 Angeles, CA, USA
 SO ARVO Annual Meeting Abstract Search and Program Planner, (2002)
 Vol. 2002,
 pp. Abstract No. 3208. cd-rom.
 Meeting Info.: Annual Meeting of the Association For Research in
 Vision
 and Ophthalmology. Fort Lauderdale, Florida, USA. May 05-10,
 2002.
 DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 26 Mar 2003

Last Updated on STN: 26 Mar 2003

L5 ANSWER 34 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2001:597119 CAPLUS

DN 135:352449

TI Inhibition of Wilms' tumor growth by intramuscular
administration of

tissue inhibitor of metalloproteinases-4 plasmid DNA

AU Celiker, M. Y.; Wang, M.; Atsidaftos, E.; Liu, X.; Liu, Y. E.;
Jiang, Y.;

Valderrama, E.; Goldberg, I. D.; Shi, Y. E.

CS Division of Pediatric Hematology/Oncology, Schneider Children's
Hospital,

Long Island Jewish Medical Center, New York, NY, 11040, USA

SO Oncogene (2001), 20(32), 4337-4343

CODEN: ONCNES; ISSN: 0950-9232

PB Nature Publishing Group

DT Journal

LA English

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 35 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2001:111082 CAPLUS

DN 134:173401

TI Tissue inhibitor of metalloproteinases-2 (TIMP-2) suppresses
TKR-growth

factor signaling independent of metalloproteinase inhibition

AU Hoegy, Susan E.; Oh, Hae-Ryong; Corcoran, Marta L.;
Stetler-Stevenson,

William G.

CS Extracellular Matrix Pathology Section, Laboratory of Pathology,
Division

of Clinical Sciences, National Institutes of Health, Bethesda,
MD,

20892-1500, USA

SO Journal of Biological Chemistry (2001), 276(5), 3203-3214

CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 36 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson
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AN 2001:258630 BIOSIS

DN PREV200100258630

TI Fibronectin upregulates TGFbeta-1 mediated TIMP-1 gene and protein

synthesis in neonatal foreskin fibroblasts.

AU Fasehun, Funmi A. [Reprint author]; Duran, Walter N. [Reprint author];

Hobson, Robert W., II; Pappas, Peter J.

CS University of Medicine and Dentistry, 185 South Orange Avenue, Newark, NJ, 07103, USA

SO FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1129. print. Meeting Info.: Annual Meeting of the Federation of American Societies for

Experimental Biology on Experimental Biology 2001. Orlando, Florida, USA.

March 31-April 04, 2001.

CODEN: FAJOEC. ISSN: 0892-6638.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 30 May 2001

Last Updated on STN: 19 Feb 2002

L5 ANSWER 37 OF 62 MEDLINE on STN

DUPLICATE 26

AN 2001447813 MEDLINE

DN PubMed ID: 11327240

TI Modulation of TIMP-1 synthesis by antiinflammatory cytokines and prostaglandin E2 in interleukin 17 stimulated human monocytes/macrophages.

AU Jovanovic D V; Di Battista J A; Martel-Pelletier J; Reboul P; He Y;

Jolicoeur F C; Pelletier J P

CS Osteoarthritis Research Unit, H pital Notre-Dame, Centre Hospitalier de

l'Universite de Montreal, Quebec, Canada.

SO Journal of rheumatology, (2001 Apr) 28 (4) 712-8.

Journal code: 7501984. ISSN: 0315-162X.

CY Canada

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200108

ED Entered STN: 20010813

Last Updated on STN: 20010813

Entered Medline: 20010809

L5 ANSWER 38 OF 62 MEDLINE on STN

DUPLICATE 27

AN 2001179938 MEDLINE

DN PubMed ID: 11168376

TI Thioredoxin alters the matrix metalloproteinase/tissue inhibitors of

metalloproteinase balance and stimulates human SK-N-SH neuroblastoma cell

invasion.

AU Farina A R; Tacconelli A; Cappabianca L; Masciulli M P; Holmgren A;
Beckett G J; Gulino A; Mackay A R

CS Section of Molecular Pathology, Department of Experimental
Medicine,
University of L'Aquila, Via Vetoio, Coppito II, 61700 L'Aquila,
Italy.

SO European journal of biochemistry / FEBS, (2001 Jan) 268 (2)
405-13.

Journal code: 0107600. ISSN: 0014-2956.

CY Germany: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200103

ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

L5 ANSWER 39 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2001:810861 CAPLUS

DN 136:33772

TI **MMP-TIMP** interaction depends on residue 2 in TIMP-4

AU Stratmann, Bernd; Farr, Martin; Tschesche, Harald

CS Biochemistry I, University of Bielefeld, Faculty of Chemistry,
Bielefeld,
D-33615, Germany

SO FEBS Letters (2001), 507(3), 285-287
CODEN: FEBLAL; ISSN: 0014-5793

PB Elsevier Science B.V.

DT Journal

LA English

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 40 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2001:82611 CAPLUS

DN 135:32045

TI Inhibition of matrix metalloproteinases by over-expression of
tissue
inhibitor of metalloproteinase-2 inhibits the growth of
experimental
hemangiomas

AU Vergani, V.; Garofalo, A.; Bani, M. R.; Borsotti, P.; Parker, M.
Pelina;
Drudis, T.; Mazzarol, G.; Viale, G.; Giavazzi, R.;
Stetler-Stevenson, W.
G.; Taraboletti, G.

CS Department of Oncology, Mario Negri Institute for Pharmacological
Research, Bergamo, 24125, Italy

SO International Journal of Cancer (2001), 91(2), 241-247

CODEN: IJCNAW; ISSN: 0020-7136

PB Wiley-Liss, Inc.

DT Journal

LA English

RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 41 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2001:34620 CAPLUS

DN 134:160811

TI Localization of the death domain of tissue inhibitor of
metalloproteinase-3 to the N terminus. Metalloproteinase
inhibition is

associated with proapoptotic activity

AU Bond, Mark; Murphy, Gillian; Bennett, Martin R.; Amour,
Augustin; Knauper,

Vera; Newby, Andrew C.; Baker, Andrew H.

CS Bristol Heart Institute, Bristol Royal Infirmary, University of
Bristol,

Bristol, BS2 8HW, UK

SO Journal of Biological Chemistry (2000), 275(52), 41358-41363

CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 42 OF 62 MEDLINE on STN

DUPLICATE 28

AN 2000175276 MEDLINE

DN PubMed ID: 10708863

TI Tissue inhibitors of metalloproteinases: evolution, structure and
function.

AU Brew K; Dinakarpanid D; Nagase H

CS Department of Biochemistry and Molecular Biology, University of
Miami

School of Medicine, Miami, FL 33101, USA..

k.brew@molbio.med.miami.edu

NC AR39189 (NIAMS)

AR40994 (NIAMS)

SO Biochimica et biophysica acta, (2000 Mar 7) 1477 (1-2) 267-83.

Ref: 74

Journal code: 0217513. ISSN: 0006-3002.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200005

ED Entered STN: 20000606

Last Updated on STN: 20000606

Entered Medline: 20000524

L5 ANSWER 43 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:597440 CAPLUS
DN 135:355780
TI Inhibition of vascular smooth muscle cell migration by eNOS gene
expression: Possible role of **MMP-2**, **MMP-9** and
TIMP-2 ratio
AU Gurjar, Milind V.; Sharma, Ram V.; Bhalla, Ramesh C.
CS Department of Anatomy and Cell Biology, University of Iowa
College of
Medicine, Iowa City, IA, 52242, USA
SO Portland Press Proceedings (2000), 16(Biology of Nitric Oxide,
Part 7),
148
CODEN: POPPEF; ISSN: 0966-4068
PB Portland Press Ltd.
DT Journal
LA English
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 44 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:486817 CAPLUS
DN 131:241239
TI Tissue inhibitor of matrix metalloproteinase-2 regulates matrix
metalloproteinase-2 activation by modulation of membrane-type 1
matrix
metalloproteinase activity in high and low invasive melanoma
cell lines
AU Kurschat, Peter; Zigrino, Paola; Nischt, Roswitha; Breitkopf,
Katja;
Steurer, Pavlos; Klein, C. Eberhard; Krieg, Thomas; Mauch,
Cornelia
CS Department of Dermatology, University of Cologne, Cologne,
50924, Germany
SO Journal of Biological Chemistry (1999), 274(30), 21056-21062
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 45 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:467036 CAPLUS
DN 131:239611
TI The specificity of TIMP-2 for matrix metalloproteinases can be
modified by
single amino acid mutations
AU Butler, Georgina S.; Hutton, Mike; Wattam, Beth A.; Williamson,
Richard

A.; Knauper, Vera; Willenbrock, Frances; Murphy, Gillian
CS School of Biological Sciences, University of East Anglia,
Norwich, NR4
7TJ, UK

SO Journal of Biological Chemistry (1999), 274(29), 20391-20396
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 46 OF 62 MEDLINE on STN DUPLICATE 29

AN 1999282197 MEDLINE

DN PubMed ID: 10353844

TI The N-terminus of collagenase **MMP-8** determines superactivity and
inhibition: a relation of structure and function analyzed by
biomolecular interaction analysis.

AU Farr M; Pieper M; Calvete J; Tschesche H

CS University of Bielefeld, Faculty of Chemistry and Biochemistry,
Germany.

SO Biochemistry, (1999 Jun 1) 38 (22) 7332-8.
Journal code: 0370623. ISSN: 0006-2960.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199906

ED Entered STN: 19990712

Last Updated on STN: 20000303

Entered Medline: 19990623

L5 ANSWER 47 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 2000:152140 CAPLUS

DN 133:68500

TI Dibutyl cyclic AMP-induced enhancement of tissue inhibitor of
metalloproteinases-3 expression and its possible relation to the
invasive

activity of the human hepatoma cell line PLC/PRF/5

AU Okamoto, Yasuyuki; Nakano, Hiroshi

CS Department of Clinico-Laboratory Diagnostics, Nara Medical
University,

Nara, 634-8521, Japan

SO Anticancer Research (1999), 19(6B), 5175-5180
CODEN: ANTRD4; ISSN: 0250-7005

PB International Institute of Anticancer Research

DT Journal

LA English

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 48 OF 62 MEDLINE on STN

DUPLICATE 30

AN 1999257345 MEDLINE
 DN PubMed ID: 10323763
 TI Role of matrix metalloproteinases and their tissue inhibitors in
 the regulation of coronary cell migration.
 AU Shi Y; Patel S; Niculescu R; Chung W; Desrochers P; Zalewski A
 CS Cardiovascular Research Center, Department of Medicine
 (Cardiology),
 Thomas Jefferson University, Philadelphia, PA 19107, USA..
 yi.shi@mail.tju.edu
 NC HL-44150 (NHLBI)
 HL-60672 (NHLBI)
 SO Arteriosclerosis, thrombosis, and vascular biology, (1999 May)
 19 (5)
 1150-5.
 Journal code: 9505803. ISSN: 1079-5642.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199906
 ED Entered STN: 19990618
 Last Updated on STN: 20000303
 Entered Medline: 19990607

L5 ANSWER 49 OF 62 MEDLINE on STN DUPLICATE 31
 AN 1999344222 MEDLINE
 DN PubMed ID: 10415733
 TI Measurement of matrix metalloproteinases and tissue inhibitors of
 metalloproteinases in blood and tissues. Clinical and
 experimental applications.
 AU Zucker S; Hymowitz M; Conner C; Zarrabi H M; Hurewitz A N;
 Matrisian L;
 Boyd D; Nicolson G; Montana S
 CS Department of Medicine and Research, Veterans Administration
 Medical Center, Northport, New York 11768, USA..
 zucker.stanley@northport.va.gov
 SO Annals of the New York Academy of Sciences, (1999 Jun 30) 878
 212-27.
 Ref: 75
 Journal code: 7506858. ISSN: 0077-8923.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 199908
 ED Entered STN: 19990820
 Last Updated on STN: 20000303

Entered Medline: 19990811

L5 ANSWER 50 OF 62 MEDLINE on STN DUPLICATE 32
AN 1999114750 MEDLINE
DN PubMed ID: 9918218
TI Interleukin 10 (IL-10) inhibition of primary human prostate
cell-induced
angiogenesis: IL-10 stimulation of tissue inhibitor of
metalloproteinase-1
and **inhibition** of matrix metalloproteinase (MMP
)-2/MMP-9 secretion.
AU Stearns M E; Rhim J; Wang M
CS Department of Pathology and Laboratory Sciences, Medical College
of
Pennsylvania and Hahnemann University, Philadelphia 19102-1192,
USA.
SO Clinical cancer research : an official journal of the American
Association
for Cancer Research, (1999 Jan) 5 (1) 189-96.
Journal code: 9502500. ISSN: 1078-0432.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990402
Last Updated on STN: 20000303
Entered Medline: 19990324

L5 ANSWER 51 OF 62 MEDLINE on STN DUPLICATE 33
AN 1999023989 MEDLINE
DN PubMed ID: 9804828
TI Differential expression of aggrecanase and matrix
metalloproteinase
activity in chondrocytes isolated from bovine and porcine
articular
cartilage.
AU Hughes C E; Little C B; Buttner F H; Bartnik E; Caterson B
CS Connective Tissue Biology Laboratories, Cardiff School of
Biosciences,
Cardiff University, Cardiff CF1 3US, Wales, United Kingdom..
hughescel@cardiff.ac.uk
SO Journal of biological chemistry, (1998 Nov 13) 273 (46)
30576-82.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 20000303

Entered Medline: 19981208

L5 ANSWER 52 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 1998:254786 CAPLUS

DN 129:53243

TI IL-10 inhibition of human prostate PC-3 ML cell metastases in SCID mice:

IL-10 stimulation of **TIMP-1** and **inhibition** of **MMP-2/MMP-9** expression

AU Stearns, M. E.; Fudge, K.; Garcia, F.; Wang, M.

CS Department of Pathology and Laboratory Medicine, Allegheny University,

Philadelphia, PA, 19102-1192, USA

SO Invasion & Metastasis (1998), Volume Date 1997, 17(2), 62-74

CODEN: INVMDJ; ISSN: 0251-1789

PB S. Karger AG

DT Journal

LA English

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 53 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN

AN 1997:345955 CAPLUS

DN 127:60267

TI Inhibition of osteolytic bone metastasis of breast cancer by combined

treatment with the bisphosphonate ibandronate and tissue inhibitor of the matrix metalloproteinase-2

AU Yoneda, Toshiyuki; Sasaki, Akira; Dunstan, Colin; Williams, Paul J.;

Bauss, Frieder; De Clerck, Yves A.; Mundy, Gregory R.

CS Department of Medicine, Division of Endocrinology and Metabolism, University of Texas Health Science Center at San Antonio, San Antonio, TX,

78284-7877, USA

SO Journal of Clinical Investigation (1997), 99(10), 2509-2517

CODEN: JCINAO; ISSN: 0021-9738

PB Rockefeller University Press

DT Journal

LA English

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 54 OF 62 MEDLINE on STN

DUPLICATE 34

AN 1998008087 MEDLINE

DN PubMed ID: 9344510

TI TIMP-3 induces cell death by stabilizing TNF-alpha receptors on the

surface of human colon carcinoma cells.

AU Smith M R; Kung H; Durum S K; Colburn N H; Sun Y

CS Intramural Research Support Program, SAIC Frederick, National Cancer

Institute, Frederick Cancer Research and Development Center,
Frederick, MD
21702, USA.

SO Cytokine, (1997 Oct) 9 (10) 770-80.
Journal code: 9005353. ISSN: 1043-4666.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199801
ED Entered STN: 19980130
Last Updated on STN: 19980130
Entered Medline: 19980122

L5 ANSWER 55 OF 62 MEDLINE on STN DUPLICATE 35
AN 1998221796 MEDLINE
DN PubMed ID: 9561025
TI IL-10 inhibition of human prostate PC-3 ML cell metastases in
SCID mice:
IL-10 stimulation of **TIMP-1** and **inhibition** of
MMP-2/MMP-9 expression.

AU Stearns M E; Fudge K; Garcia F; Wang M
CS Department of Pathology and Laboratory Medicine, Allegheny
University,
Philadelphia, Pa. 19102-1192, USA.

SO Invasion & metastasis, (1997) 17 (2) 62-74.
Journal code: 8202435. ISSN: 0251-1789.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980514
Last Updated on STN: 20000303
Entered Medline: 19980505

L5 ANSWER 56 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:177079 CAPLUS
DN 128:293277
TI Tissue inhibitors of metalloproteinases and metastasis
AU Iwata, Hiroji; Kobayashi, Syunzo
CS Second Dep. Surg., Nagoya City Univ. Sch. Med., Japan
SO Ketsueki, Men'eki, Shuyo (1996), 1(4), 348-355
CODEN: KMSHF6; ISSN: 1341-5824
PB Medikaru Rebyusha
DT Journal; General Review
LA Japanese

L5 ANSWER 57 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1996:648357 CAPLUS
DN 126:58732
TI Interleukin-4 blocks the release of collagen fragments from
bovine nasal

cartilage treated with cytokines
 AU Cawston, Tim E.; Ellis, Alison J.; Bigg, Heather; Curry,
 Valerie; Lean,
 Eileen; Ward, Dawn
 CS Rheumatology Research Unit, Box 194, Addenbrooke's Hospital,
 Cambridge,
 CB2 2QQ, UK
 SO Biochimica et Biophysica Acta, Molecular Cell Research (1996),
 1314(3),
 226-232
 CODEN: BBAMCO; ISSN: 0167-4889
 PB Elsevier B.V.
 DT Journal
 LA English

L5 ANSWER 58 OF 62 MEDLINE on STN DUPLICATE 36
 AN 95355479 MEDLINE
 DN PubMed ID: 7629179
 TI Steps involved in activation of the pro-matrix metalloproteinase
 9
 (progelatinase B)-tissue inhibitor of metalloproteinases-1
 complex by
 4-aminophenylmercuric acetate and proteinases.
 AU Ogata Y; Itoh Y; Nagase H
 CS Department of Biochemistry and Molecular Biology, University of
 Kansas
 Medical Center, Kansas City 66160-7421, USA.
 NC AR39189 (NIAMS)
 AR40994 (NIAMS)
 SO Journal of biological chemistry, (1995 Aug 4) 270 (31) 18506-11.
 Journal code: 2985121R. ISSN: 0021-9258.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199509
 ED Entered STN: 19950921
 Last Updated on STN: 20000303
 Entered Medline: 19950907

L5 ANSWER 59 OF 62 MEDLINE on STN DUPLICATE 37
 AN 95301511 MEDLINE
 DN PubMed ID: 7782289
 TI The gene structure of tissue inhibitor of metalloproteinases
 (TIMP)-3 and
 its inhibitory activities define the distinct TIMP gene family.
 CM Erratum in: J Biol Chem. 1996 Feb 2;271(5):2874. PubMed ID:
 8576269
 AU Apte S S; Olsen B R; Murphy G
 CS Department of Cell Biology, Harvard Medical School, Boston,
 Massachusetts

02115, USA.

NC AR36819 (NIAMS)
AR36820 (NIAMS)
HL33014 (NHLBI)
+

SO Journal of biological chemistry, (1995 Jun 16) 270 (24) 14313-8.

Journal code: 2985121R. ISSN: 0021-9258.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

OS GENBANK-U26433; GENBANK-U26434; GENBANK-U26435; GENBANK-U26436;
GENBANK-U26437

EM 199507

ED Entered STN: 19950726
Last Updated on STN: 20000303
Entered Medline: 19950717

L5 ANSWER 60 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson
Corporation on
STN

AN 1995:97255 BIOSIS

DN PREV199598111555

TI Role of **TIMP** and **MMP inhibition** in
preventing connective tissue breakdown.

AU Cawston, Tim; Plumpton, Tracy; Curry, Valerie; Ellis, Alison;
Powell, Liz

CS Rheumatology Res. Unit, Box 194, Addenbrookes Hosp., Hill's Road,
Cambridge CB2 2QQ, UK

SO Greenwald, R. A. [Editor]; Golub, L. M. [Editor]. Ann. N. Y.
Acad. Sci.,
(1994) pp. 75-83. Annals of the New York Academy of Sciences;
Inhibition
of matrix metalloproteinases: Therapeutic potential.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New
York, New
York 10021, USA. Series: Annals of the New York Academy of
Sciences.
Meeting Info.: Conference. Tampa, Florida, USA. January 19-22,
1994.
CODEN: ANYAA9. ISSN: 0077-8923. ISBN: 0-89766-900-2 (paper),
0-89766-899-5
(cloth).

DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)

LA English

ED Entered STN: 1 Mar 1995
Last Updated on STN: 1 Mar 1995

L5 ANSWER 61 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:313189 CAPLUS
DN 123:4253
TI Characterization of tissue inhibitor of metalloproteinases 2
(TIMP-2) from
human hepatoma cell line (HLE cells) and its interaction with
gelatinase A
and inactivation
AU Kawamura, Kouji
CS School Medicine, Kanazawa Univ., Kanazawa, 920, Japan
SO Kanazawa Daigaku Juzen Igakkai Zasshi (1994), 103(4), 690-700
CODEN: JUZIAG; ISSN: 0022-7226
PB Juzen Igakkai
DT Journal
LA Japanese

L5 ANSWER 62 OF 62 MEDLINE on STN DUPLICATE 38
AN 95069560 MEDLINE
DN PubMed ID: 7978853
TI Role of **TIMP** and **MMP inhibition** in
preventing connective tissue breakdown.
AU Cawston T; Plumpton T; Curry V; Ellis A; Powell L
CS Rheumatology Research Unit, Addenbrookes Hospital, Cambridge,
United
Kingdom.
SO Annals of the New York Academy of Sciences, (1994 Sep 6) 732
75-83.
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199412
ED Entered STN: 19950110
Last Updated on STN: 20000303
Entered Medline: 19941202

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research documents
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conditions
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NEWS 9 OCT 04 CA/CAPplus-Canadian Intellectual Property Office
(CIPO) added
to core patent offices
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NEWS 11 OCT 13 New CAS Information Use Policies Effective October
17, 2005
NEWS 12 OCT 17 STN(R) AnaVist(TM), Version 1.01, allows the
export/download
of CAPplus documents for use in third-party analysis
and
visualization tools
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NEWS 14 OCT 27 DIOGENES content streamlined
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research

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(4A)(inhibition or inhibit or inhibiting or inhibited)
L1 1846 (TIMP OR (TISSUE INHIBITORS OF METALLOPROTEINASES))
(4A)(INHIBIT
ION OR INHIBIT OR INHIBITING OR INHIBITED)

=> s ((Matrix metalloproteinase) or MMP)(4A)(inhibition or inhibit
or inhibiting or inhibited)
L2 8293 ((MATRIX METALLOPROTEINASE) OR MMP)(4A)(INHIBITION OR
INHIBIT
OR INHIBITING OR INHIBITED)

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L3 669 L1 AND L2

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L4 23 L3 AND (2001 OR 2000 OR 1999 OR 1998)

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L5 ANSWER 1 OF 15 MEDLINE on STN DUPLICATE 1

AN 2004509808 MEDLINE

DN PubMed ID: 15467768

TI An immunohistochemical study of TIMP-3 expression in oesophageal
squamous
cell carcinoma.

AU Miyazaki T; Kato H; Nakajima M; Faried A; Takita J; Sohda M;
Fukai Y;

Yamaguchi S; Masuda N; Manda R; Fukuchi M; Ojima H; Tsukada K;
Kuwano H

CS Department of General Surgical Science (Surgery I), Gunma
University

Graduate School of Medicine, 3-39-22, Showa-machi, Maebashi,
Gunma

371-8511, Japan.. tatsuyam@showa.gunma-u.ac.jp

SO British journal of cancer, (2004 Oct 18) 91 (8) 1556-60.

Journal code: 0370635. ISSN: 0007-0920.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200411

ED Entered STN: 20041014

Last Updated on STN: 20041117

Entered Medline: 20041116

AB Tissue inhibitor of metalloproteinase-3 (**TIMP-3**)

inhibits the activity of **matrix**

metalloproteinase, which may play an important role in carcinoma
invasion and metastasis. We have investigated the relationship

between

TIMP-3 reduction and clinicopathological factors in oesophageal
squamous

cell carcinoma (ESCC). We examined tissue specimens that had
been removed

from 90 patients with thoracic oesophageal cancer who had
undergone

surgery between 1983 and 2001. Immunohistochemical staining was

performed by the standard streptavidin-biotin method.
Immunostaining of
TIMP-3 was seen in the cytoplasm of cancer cells and normal
oesophageal
epithelial cells, particularly in cells located in shallow areas
of the
tumour. TIMP-3 preserved (+), moderate (+/-), and reduced (-)
cases
accounted for 30, 27, and 33 of the 90 patients, respectively
(33, 30,
37%). Significant correlations were observed between TIMP-3
expression
and depth of tumour invasion ($P=0.001$), number of lymph node
metastases
($P=0.003$), infiltrative growth pattern ($P=0.003$), and disease
stage
($P=0.005$). The survival rates of patients with TIMP-3 (-)
cancer were
significantly lower than those of patients with TIMP-3 (+) and
TIMP-3
(+/-) cancer ($P=0.0003$). The mean 5-year survival rates of
patients with
TIMP-3 (+), (+/-), and (-) were 50, 58, and 21%, respectively.
In
conclusion, decreased expression of TIMP-3 protein correlates
with
invasive activity and metastasis. This makes the prognosis for
patients
with cancer that has lost TIMP-3 significantly less favourable
than that
for patients with cancer that has maintained TIMP-3.

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AN 2002314329 EMBASE

TI Dexamethasone inhibits vascular smooth muscle cell migration via
modulation of matrix metalloproteinase activity.

AU Pross C.; Farooq M.M.; Angle N.; Lane J.S.; Cerveira J.J.;
Xavier A.E.;

Freischlag J.A.; Law R.E.; Gelabert H.A.

CS Dr. H.A. Gelabert, Medical Plaza 200, Los Angeles, CA 90095,
United

States. hgelaert@mednet.ucla.edu

SO Journal of Surgical Research, (2002) Vol. 102, No. 2, pp. 57-62.
Refs: 28

ISSN: 0022-4804 CODEN: JSGRA2

CY United States

DT Journal; Article

FS 009 Surgery

018 Cardiovascular Diseases and Cardiovascular Surgery

030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 20020919

Last Updated on STN: 20020919

AB Background. Dexamethasone (DEX) has been shown to inhibit development of

neointimal hyperplasia in rats. We hypothesize that DEX **inhibits** neointimal hyperplasia by altering **matrix**

metalloproteinase (MMP) activity, resulting in

inhibition of smooth muscle cell migration. Methods. Rat aortic smooth muscle cells (RASMC) were harvested and cultured for two

to four

passages. A migration assay was performed in a Boyden chamber

with

chemoattractant (platelet-derived growth factor) and varying concentrations of DEX (10^{-9} to 10^{-5} M). The number of

migrated cells

was counted under light microscopy. Zymography was performed on culture

media to assess MMP activity, and Western blotting was performed to assay

MMP and levels of tissue inhibitors of MMPs (TIMPs). Results.

DEX

progressively inhibited RASMC migration in a dose-dependent fashion. This

effect was statistically significant for concentrations of 10^{-7} to

10^{-5} M ($P < 0.0005$). Zymography showed that DEX **inhibits**

MMP-2 activity in a dose-dependent manner. Western blots

indicated that total **MMP-2** secretion was **inhibited** and

that **TIMP-2** secretion was increased by DEX. Conclusions. DEX

inhibits platelet-derived growth factor-induced migration of

RASMCs and

MMP-2 activity in vitro. Our data suggest that DEX suppresses

MMP

activity and secretion, resulting in the inhibition of smooth muscle cell

migration. This may explain the mechanism by which DEX inhibits neointimal hyperplasia. .COPYRGT. 2001 Elsevier Science.

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AN 2001076223 EMBASE

TI Effects of nitric oxide on matrix metalloproteinase-2 production by

rheumatoid synovial cells.

AU Hirai Y.; Migita K.; Honda S.; Ueki Y.; Yamasaki S.; Urayama S.; Kamachi

M.; Kawakami A.; Ida H.; Kita M.; Fukuda T.; Shibatomi K.; Kawabe Y.;

Aoyagi T.; Eguchi K.
 CS K. Eguchi, First Dept. of Internal Medicine, Nagasaki Univ.
 School of
 Medicine, Sakamoto 1-7-1, Nagasaki 852-8501, Japan
 SO Life Sciences, (12 Jan 2001) Vol. 68, No. 8, pp. 913-920.
 Refs: 22
 ISSN: 0024-3205 CODEN: LIFSAK
 PUI S 0024-3205(00)00998-X
 CY United States
 DT Journal; Article
 FS 005 General Pathology and Pathological Anatomy
 031 Arthritis and Rheumatism
 LA English
 SL English
 ED Entered STN: 20010316
 Last Updated on STN: 20010316
 AB Nitric oxide (NO) is a multifunctional messenger molecule
 generated from
 L-arginine by a family of enzymes, including nitric oxide
 synthase (NOS).
 This study was performed to examine whether NO modulates the
 production of
 matrix metalloproteinases (MMPs), which degrade all components of
 extracellular matrix (ECM), in rheumatoid synovial cells. We
 investigated
 the effects of exogenously generated NO by a NO donor,
 S-nitroso-N-acetyl-DL-penicillamine (SNAP), on the MMPs
 production by
 rheumatoid synovial cells. Culture media conditioned by
 SNAP-treated
 synovial cells were examined by gelatin zymography and immunoblot
 analysis. Incubation of synovial cells with SNAP resulted in
 gelatinase A
 production in a dose-dependent fashion. Furthermore, RT-PCR
 analysis
 demonstrated that MMP-2 mRNA expression was induced in
 SNAP-treated
 synovial cells. In contrast, SNAP did not influence the
 production of
 TIMP-1 and **TIMP-2**, which preferentially **inhibit**
MMP-2, by rheumatoid synovial cells. Our data indicate that NO
 could modulate MMP production by rheumatoid synovial cells and
 therefore
 contribute to ECM degradation of articular components in RA.
 .COPYRGHT.
 2001 Elsevier Science Inc.

L5 ANSWER 4 OF 15 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All
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 reserved on STN
 AN 2001240567 EMBASE
 TI High glucose decreases matrix metalloproteinase-2 activity in rat

mesangial cells via transforming growth factor- β (1).

AU Singh R.; Ruo Hua Song; Alavi N.; Pegoraro A.A.; Singh A.K.;
Leehey D.J.

CS Dr. D.J. Leehey, Veterans Affairs Hospital, Hines, IL 60141,
United

States. david.leehey@med.va.gov

SO Experimental Nephrology, (2001) Vol. 9, No. 4, pp. 249-257.

Refs: 32

ISSN: 1018-7782 CODEN: EXNEEG

CY Switzerland

DT Journal; Article

FS 003 Endocrinology

028 Urology and Nephrology

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20010726

Last Updated on STN: 20010726

AB Diabetic nephropathy is characterized by accumulation of
mesangial matrix.

Glucose-induced inhibition of matrix-degrading enzymes such as
collagenases is believed to contribute to matrix accumulation.

We have

previously demonstrated that 72 kDa type IV collagenase activity

is

decreased in the rat mesangial cells cultured in high glucose

media

[Diabetes 1995;44:929-935]. The present studies were designed to
investigate if the cytokine transforming growth factor- β (1)

(TGF- β (1)) mediates this effect of glucose. Type IV

collagenases

degrade type IV collagen as well as gelatin (denatured collagen)

and are

thus also called gelatinases. They belong to the family of

matrix

metalloproteinases (MMPs); MMP activity is controlled by tissue

inhibitors

of metalloproteinases (TIMPs). The activity of 72 kDa type IV

collagenase, also known as matrix metalloproteinase-2 (MMP-2),

was

assessed using three methods: (1) fluoresceinated gelatin

degradation

assay to detect free enzyme activity (activity which is present

in excess

of **TIMP-inhibited** activity); (2) zymography to measure

total (free + TIMP-bound) enzyme activity; (3) ELISA using

specific

antibodies to measure MMP-2 levels. TGF- β (1) and TIMP-2 levels

were)also determined by ELISA. Incubation of primary cultures

of rat

mesangial cells for 5 days in 30 vs. 5 mM glucose resulted in a

3-fold

increase in production of total TGF- β (1), a significant decrease in MMP-2 activity and immunoreactive MMP-2 levels, and an increase in TIMP-2 levels. Addition of exogenous TGF- β (1) to mesangial cells incubated in 5 mM glucose replicated the high glucose effect by producing a significant decrease in MMP-2 levels with a concurrent increase in TIMP-2 levels. Furthermore, glucose-induced **inhibition** of MMP-2 activity was completely blocked by neutralization of TGF- β (1) with anti-TGF- β (1) antibody. We conclude that the decrease in MMP-2 activity induced by glucose loading is mediated via TGF- β (1).
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AN 2001016268 EMBASE

TI **Inhibition of matrix metalloproteinases** by over-expression of tissue inhibitor of metalloproteinase-2 inhibits the growth of experimental hemangiomas.

AU Vergani V.; Garofalo A.; Bani M.R.; Borsotti P.; Parker M.P.; Drudis T.;

Mazzarol G.; Viale G.; Giavazzi R.; Stetler-Stevenson W.G.; Taraboletti G.

CS G. Taraboletti, Department of Oncology, Mario Negri Inst. of Pharmacol.

Res., via Gavazzeni 11, 24125 Bergamo, Italy.

taraboletti@irfmm.mnegri.it

SO International Journal of Cancer, (15 Jan 2001) Vol. 91, No. 2, pp.

241-247.

Refs: 39

ISSN: 0020-7136 CODEN: IJCNAAW

CY United States

DT Journal; Article

FS 016 Cancer

030 Pharmacology

037 Drug Literature Index

LA English

SL English

ED Entered STN: 20010125

Last Updated on STN: 20010125

AB Inhibitors of proteases prevent tumor-associated matrix degradation,

affecting tumor growth, angiogenesis and metastasis. Our study was

designed to investigate the effect of **inhibition** of **matrix metalloproteinases (MMPs)** on the growth

of experimental hemangiomas, using the model of murine endothelioma eEnd.1 cells. In nude mice, these cells generate hemangiomas, consisting mostly of host-recruited endothelial cells, whose growth requires the activity of MMPs. In vitro, eEnd.1 cells produce factors that recruit endothelial cells and stimulate them to release MMPs. Over-expression of TIMP-2, following retrovirus-mediated gene transfer, decreased tumor growth in vivo. The infected clone CR1, which produces high levels of TIMP-2 (as assessed by Northern blot, ELISA and reverse zymography), formed slow-growing tumors that did not grow beyond 0.4 g, while clone IH, which produces little TIMP-2, grew not dissimilarly to mock-infected cells and parental e.End.1 cells. Histologically, control tumors presented the features of cavernous hemangiomas, while CR1 tumors had a more solid pattern, showing foci of apoptotic cells. In vitro, TIMP-2 over-expression had no autocrine anti-proliferative effect on endothelioma cells but reduced their ability to recruit endothelial cells. CR1 cells lacked the capacity of mock-infected or parental eEnd.1 cells to stimulate endothelial cell motility and invasiveness. Antibodies against TIMP-2 restored the ability of CR1 to induce endothelial cell invasion. We conclude that, in this model, genetic increase of **TIMP-2 inhibits** tumor growth, apparently by affecting the recruitment and organization of host endothelial cells by the transformed cells.

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DUPLICATE 2

AN 2001246293 EMBASE

TI CD40L induces matrix-metalloproteinase-9 but not tissue inhibitor of

metalloproteinases-1 in cervical carcinoma cells: Imbalance between

NF- κ B and STAT3 activation.

AU Smola-Hess S.; Schnitzler R.; Hadaschik D.; Smola H.; Mauch C.; Krieg T.;

Pfister H.

CS S. Smola-Hess, Institute of Virology, University of Cologne,
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SO Experimental Cell Research, (15 Jul 2001) Vol. 267, No. 2, pp.
205-215.

Refs: 76
ISSN: 0014-4827 CODEN: ECREAL

CY United States

DT Journal; Article

FS 005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20010726
Last Updated on STN: 20010726

AB Matrix-metalloproteinases (MMPs) are essentially required for
tumor cell
invasion and metastasis. Production of precursor enzymes is
regulated on
transcriptional level, while activation of the pro-enzymes is
tightly
controlled by posttranscriptional mechanisms. The enzyme
activity can be
blocked by specific tissue inhibitors of MMPs (TIMPs). In
cervical
carcinomas strong up-regulation of the type IV collagenase MMP-9
had been
demonstrated. We show that activation of CD40, a receptor highly
expressed on cervical carcinomas, induces MMP-9 in cervical
carcinoma
cells, whereas **TIMP-1** production **inhibiting**
MMP-9 activity was not affected. This gene induction pattern
corresponded to the differential activation of the transcription
factor
nuclear factor κ B (NF- κ B) regulating MMP-9, but not signal
transducer and activator of transcription 3 (STAT3), which is
involved in
TIMP-1 gene regulation. Transient expression of the
CD40-inducible
NF- κ B subunit p65 was sufficient for MMP-9 induction. Agents
that
suppressed CD40-mediated NF- κ B activation also reduced MMP-9
induction, further supporting an important role of NF- κ B in
CD40-mediated MMP-9 induction. Our data suggest that CD40
expression in
carcinoma cells might convert a CD40L-dependent immunological
defense
signal into a tumor-promoting signal. Selective CD40-mediated
signaling
through NF- κ B but not STAT3 correlates to a shift of the balance
between MMP-9 and TIMP-1 toward the protease. .COPYRGHT. 2001
Academic Press.

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DUPLICATE 3

AN 2001195366 EMBASE

TI Type I collagen stabilization of matrix metalloproteinase-2.

AU Ellerbroek S.M.; Wu Y.I.; Stack M.S.

CS M.S. Stack, Northwestern Univ. Medical School, Department of Cell, 303 E.

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SO Archives of Biochemistry and Biophysics, (1 Jun 2001) Vol. 390, No. 1, pp.

51-56.

Refs: 35

ISSN: 0003-9861 CODEN: ABBIA4

CY United States

DT Journal; Article

FS 029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20010614

Last Updated on STN: 20010614

AB The activity of matrix metalloproteinase-2 (MMP-2) is regulated stringently on the posttranslational level. MMP-2 efficiently undergoes

autolysis into inactive polypeptides in vitro, prompting the hypothesis

that MMP-2 autolysis may function as an alternative mechanism for posttranslational control of MMP-2 in vivo. Moreover, MMP-2 binds to

intact type I collagen fibrils; however, the functional consequences of

this interaction have not been fully elucidated. To test the hypothesis

that MMP-2 binding to type I collagen functions as a positive regulator of

MMP-2 proteolytic potential, the effect of type I collagen on MMP-2 activity, **inhibition** by tissue inhibitor of metalloproteinase-2 (TIMP-2), and enzyme stability was examined.

Here, we

report that purified MMP-2 binds but does not cleave intact type

I

collagen. The presence of type I collagen affects neither enzymatic

activity against a quenched fluorescent peptide substrate nor the kinetics

of **inhibition** by TIMP-2. However, MMP-2 is stabilized

from autolysis in the presence of type I collagen, but not by elastin,

fibrinogen, or laminin. These data provide biochemical evidence that

MMP-2 exosite interactions with type I collagen may function in the posttranslational control of MMP-2 activity by reducing the rate of autolytic inactivation. .COPYRGT. 2001 Academic Press.

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DUPLICATE 4

AN 2000228169 EMBASE

TI Regulation and significance of hepatocyte-derived matrix metalloproteinases in liver remodeling.

AU Haruyama T.; Ajioka I.; Akaike T.; Watanabe Y.

CS Y. Watanabe, Department Biomolecular Engineering, Tokyo Institute of

Technology, 4259 Nagatsuda, Midori-ku, Yokohama 226-850, Japan.
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SO Biochemical and Biophysical Research Communications, (16 Jun 2000) Vol.

272, No. 3, pp. 681-686.

Refs: 36

ISSN: 0006-291X CODEN: BBRCA

CY United States

DT Journal; Article

FS 029 Clinical Biochemistry
048 Gastroenterology

LA English

SL English

ED Entered STN: 20000720

Last Updated on STN: 20000720

AB Regulation in expression and activation of proteinases is one of the most

important mechanisms in organ morphogenesis. In this study, we investigated the expression of MMPs in primary hepatocytes and their roles

in liver remodeling. A hepatocyte proliferation initiating cytokine,

TNF α , induced MMP-9 expression in these cells while the expression

of MMP-2 did not change by zymography analysis. Interestingly, both the

induced MMP-9 expression and hepatocyte proliferation by TNF α were

synergistically enhanced by HGF in vitro. The increased proliferation was

suppressed by MMP inhibitor TIMP-1, suggesting that cytokine-induced MMP

regulates proliferation. The increased expression of MMP-9 by the

cytokines was inhibited by cytochalasin D or colchicine but not by PI3

kinase inhibitor wortmannin. In addition, costimulation by TNF α and

HGF of spheroidal hepatocytes cultured in 3-dimensional collagen gel drastically induced morphological changes by cell extension and migration in the gel, which was in parallel with the induced expression of MMP-9 and was inhibited by TIMP-1 and -2. The MMP activity was also detected in vivo in the regenerating liver after partial hepatectomy by in situ zymography. These results suggest the roles of MMPs produced by parenchymal cells in liver remodeling.

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L5 ANSWER 9 OF 15 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights

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AN 2000383693 EMBASE

TI Prognostic impact of tissue inhibitor of matrix metalloproteinase-1 in esophageal carcinoma.

AU Mori M.; Mimori K.; Sadanaga N.; Inoue H.; Tanaka Y.; Mafune K.-I.; Ueo H.; Barnard G.F.

CS M. Mori, Department of Surgery, Medical Institute of Bioregulation, Kyushu University, 4546 Tsurumibaru, Beppu 874-0838, Japan.

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SO International Journal of Cancer, (2000) Vol. 88, No. 4, pp. 575-578.

Refs: 27

ISSN: 0020-7136 CODEN: IJCNW

CY United States

DT Journal; Article

FS 005 General Pathology and Pathological Anatomy

011 Otorhinolaryngology

016 Cancer

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20001213

Last Updated on STN: 20001213

AB Tissue inhibitor of metalloproteinase-1 (TIMP-1)

inhibits the activity of matrix

metalloproteinase, which may play an important role in carcinoma invasion and metastasis. TIMP-1 is thus considered to inhibit carcinoma

invasion and metastasis. However, TIMP-1 possesses another important

function, cell growth promotion. The clinical significance of TIMP-1

expression has not been fully determined in esophageal carcinoma. We thus examined the expression of TIMP-1 mRNA in tumor (T) and corresponding normal (N) tissues of 85 esophageal carcinoma cases by RT-PCR. The T:N ratio of TIMP-1 mRNA expression in each case was evaluated semiquantitatively with adjustment by an internal control gene. The mean T:N ratio was 2.0 (range 0.2-6.5). When comparing high-expression cases (T:N > 2.0, n = 37) with low-expression cases (T:N ≤ 2.0, n = 48), the former showed a significantly higher frequency of lymph vessel invasion, vascular vessel invasion, lymph node metastasis and advanced-stage disease. The former cases showed a poorer prognosis than the latter. Multivariate analysis disclosed that TIMP-1 expression status was an independent determining factor for prognosis. Our findings suggest that TIMP-1 expression correlates with tumor extension of esophageal carcinoma and might, if validated, prove useful as a novel prognostic marker for esophageal carcinoma. (C) 2000 Wiley-Liss, Inc.

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AN 2000281993 EMBASE

TI Regulation of tissue inhibitor of metalloproteinase-3 (Timp-3) mRNA

expression during rat CNS development.

AU Jaworski D.M.; Fager N.

CS Dr. D.M. Jaworski, Dept. of Anatomy/Neurobiology, Univ. of Vermont College

of Medicine, Given C454, Burlington, VT 05405, United States. djaworsk@zoo.uvm.edu

SO Journal of Neuroscience Research, (15 Aug 2000) Vol. 61, No. 4, pp.

396-408.

Refs: 77

ISSN: 0360-4012 CODEN: JNREDK

CY United States

DT Journal; Article

FS 008 Neurology and Neurosurgery
029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20000831

Last Updated on STN: 20000831

AB To preserve tissue integrity during the structural rearrangements that occur during central nervous system (CNS) development, an intricate balance between extra-cellular matrix (ECM) synthesis and degradation must be maintained. The matrix metalloproteinases (MMPs) are believed to be the main mediators of ECM degradation. Because MMPs function in the turnover of a broad-spectrum of ECM proteins their activity is tightly regulated by interaction with tissue inhibitors of metalloproteinases (TIMPs). Whereas the primary function of **TIMPs** is to **inhibit MMP** activity, evidence is mounting that TIMPs are multifunctional molecules that exert diverse cell biological functions distinct from their MMP-inhibitory activities. Although the role of MMPs and TIMPs in the morphogenesis of non-neural tissues has been investigated, to date few studies have analyzed MMP or TIMP expression during CNS development. In the present report, we demonstrate the regulation of Timp-3 mRNA expression throughout the course of CNS development. In particular, Timp-3 mRNA is expressed in embryonic ventricular zones and the postnatal subventricular zone (SVZ). In addition, Timp-3 is expressed in the rostral migratory stream (RMS) to the olfactory bulb in a pattern similar to the ECM proteoglycan brevican. These data suggest that TIMP-3 and brevican may act in concert to guide neuronal migration along the RMS. (C) 2000 Wiley-Liss, Inc.

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AN 2000081145 EMBASE

TI Tissue inhibitors of metalloproteinases: Evolution, structure and function.

AU Brew K.; Dinakarpandian D.; Nagase H.

CS K. Brew, Department of Biochemistry, University of Miami, School of

Medicine, Miami, FL 33101, United States.

kbrew@molbio.med.miami.edu

SO Biochimica et Biophysica Acta - Protein Structure and Molecular Enzymology, (7 Mar 2000) Vol. 1477, No. 1-2, pp. 267-283.

Refs: 74
 ISSN: 0167-4838 CODEN: BBAEDZ
 PUI S 0167-4838(99)00279-4
 CY Netherlands
 DT Journal; General Review
 FS 029 Clinical Biochemistry
 LA English
 SL English
 ED Entered STN: 20000316
 Last Updated on STN: 20000316
 AB The matrix metalloproteinases (MMPs) play a key role in the normal physiology of connective tissue during development, morphogenesis and wound healing, but their unregulated activity has been implicated in numerous disease processes including arthritis, tumor cell metastasis and atherosclerosis. An important mechanism for the regulation of the activity of MMPs is via binding to a family of homologous proteins referred to as the tissue inhibitors of metalloproteinases (TIMP-1 to TIMP-4). The two-domain TIMPs are of relatively small size, yet have been found to exhibit several biochemical and physiological/biological functions, including **inhibition** of active **MMPs**, proMMP activation, cell growth promotion, matrix binding, inhibition of angiogenesis and the induction of apoptosis. Mutations in TIMP-3 are the cause of Sorsby's fundus dystrophy in humans, a disease that results in early onset macular degeneration. This review highlights the evolution of TIMPs, the recently elucidated high-resolution structures of TIMPs and their complexes with metalloproteinases, and the results of mutational and other studies of structure-function relationships that have enhanced our understanding of the mechanism and specificity of the **inhibition** of **MMPs** by **TIMPs**. Several intriguing questions, such as the basis of the multiple biological functions of TIMPs, the kinetics of TIMP-MMP interactions and the differences in binding in some TIMP-metalloproteinase pairs are discussed which, though not fully resolved, serve to illustrate the kind of issues that are important for a full understanding of the interactions between families of molecules.

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AN 2000434576 EMBASE

TI TIMP-1/MMP-9 imbalance in an EBV-immortalized B lymphocyte cellular model:

Evidence for TIMP-1 multifunctional properties.

AU Gaudin P.; Trocme C.; Berthier S.; Kieffer S.; Boutonnat J.; Lamy C.;

Surla A.; Garin J.; Morel F.

CS F. Morel, GREPI, Laboratoire d'Enzymologie, CHU A. Michallon, BP 217,

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SO Biochimica et Biophysica Acta - Molecular Cell Research, (11 Dec 2000)

Vol. 1499, No. 1-2, pp. 19-33.

Refs: 46

ISSN: 0167-4889 CODEN: BAMRDP

PUI S 0167-4889(00)00084-7

CY Netherlands

DT Journal; Article

FS 026 Immunology, Serology and Transplantation

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20010119

Last Updated on STN: 20010119

AB Tissue inhibitors of metalloproteinases (TIMPs) were initially described

as agents controlling metalloproteinase activity. The purpose of this

study was to investigate the expression and the roles of TIMP-1 secreted

by Epstein-Barr-virus (EBV)-immortalized B lymphocytes. TIMP-1 was

isolated from conditioned medium of interleukin (IL)-1 β stimulated

EBV-B lymphocytes; purified TIMP-1 was identified by mass spectrometry and

immunochemistry. TIMP-1-free MMP-9 was quantified after purification by

zymography and enzyme-linked immunosorbent assay. EBV-B lymphocyte-secreted **TIMP-1 inhibited MMP-9**

gelatinolytic activity resulting in decreased B-cell transmigration as

measured in vitro. The release of huge amounts of TIMP-1 in proportion to

MMP-9 from B lymphocytes after EBV transformation was shown to be correlated with secretion of IL-10 and dependent on culture time. In

contrast, there was little TIMP-1 and almost no IL-10 released from native

B cells, suggesting a possible IL-10 mediated autocrine regulation

mechanism of TIMP-1 synthesis. The MMP-9/TIMP-1 imbalance observed in the

culture medium of EBV-B lymphocytes (TIMP-1>MMP-9) and of native B cells

(MMP-9>TIMP-1) is suggestive of a new function for TIMP-1. We propose

that TIMP-1 acts as a survival factor controlling B-cell growth and

apoptosis through an autocrine regulation process involving IL-10 secreted

by EBV-B lymphocytes. (C) 2000 Elsevier Science B.V.

L5 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN

AN 1999:196812 CAPLUS

DN 131:97052

TI Inhibition of Endothelial Cell Migration by Gene Transfer of Tissue

Inhibitor of Metalloproteinases-1

AU Fernandez, Harold A.; Kallenbach, Klaus; Seghezzi, Graziano; Grossi,

Eugene; Colvin, Stephen; Schneider, Robert; Mignatti, Paolo; Galloway,

Aubrey

CS Department of Surgery, NYU Medical Center, Section of Cardiothoracic

Surgery, New York, NY, 10016, USA

SO Journal of Surgical Research (1999), 82(2), 156-162

CODEN: JSGRA2; ISSN: 0022-4804

PB Academic Press

DT Journal

LA English

AB Angiogenesis requires degradation of the vessel's basal lamina and endothelial

cell migration into the tissue stroma. Matrix metalloproteinases (MMPs)

and their tissue inhibitors (TIMPs) play important roles in this process.

MMP activity is tightly regulated during vessel growth. This work was

designed to characterize the effect of TIMP-1 upregulation on endothelial

cell invasion of the extracellular matrix. We constructed replication-deficient recombinant adenoviruses that encode

either TIMP-1

(Ad.TIMP-1) or Escherichia coli lac Z (Ad. β gal) cDNA. Bovine aortic

endothelial (BAE) cells were infected with 100 infectious particles/cell.

Gene expression was assessed by Northern and Western blotting. TIMP-1 activity in cell-conditioned media was measured by a resorufin-labeled casein protease assay. BAE cell migration was measured by Boyden chamber assays with 0.2% gelatin-coated, 8.0- μ m polycarbonate membranes. Results. TIMP-1 was overexpressed by Ad.TIMP-1-infected BAE cells relative to control, Ad. β gal-infected or uninfected cells. TIMP-1 activity in Ad.TIMP-1 cell-conditioned medium was 2.8-fold higher than in control cells. By Boyden chamber assays with gelatin-coated membranes, Ad.TIMP-1-infected BAE cells showed $89.97 \pm 1.64\%$ (mean \pm SEM) reduction in migration relative to Ad. β gal-infected cells ($P < 0.02$) and $90.53 \pm 1.12\%$ relative to uninfected cells ($P < 0.02$). Without gelatin coating, migration was equivalent in all groups. The replication-deficient recombinant adenovirus we constructed affords rapid and efficient upregulation of functional TIMP-1 in endothelial cells. Infection results in a dramatic decrease in cell migration and invasion of extracellular matrix. Thus, such a recombinant vector may provide a useful tool for the gene therapy of vascular remodeling and inhibition of angiogenesis. (c) 1999 Academic Press.

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:521650 CAPLUS
DN 129:275230
TI Short chain fatty acids inhibit human (SW1116) colon cancer cell invasion by reducing urokinase plasminogen activator activity and stimulating TIMP-1 and TIMP-2 activities, rather than via MMP modulation
AU Emenaker, Nancy J.; Basson, Marc D.
CS Department of Surgery, Yale University School of Medicine, New Haven, CT, 06520-8062, USA
SO Journal of Surgical Research (1998), 76(1), 41-46
CODEN: JSGRA2; ISSN: 0022-4804
PB Academic Press
DT Journal

LA English
AB Short-chain fatty acids (SCFA) derived from dietary fiber may protect against invasive colon cancer by modulating degradative matrix metalloproteinases (MMPs) and protective tissue inhibitor of metalloproteinases (TIMPs). Since the invasion depends on the MMP/TIMP ratio, SCFA may inhibit the colon cancer invasion by inhibiting MMPs and stimulating TIMPs. SW1116 colon cancer cells were seeded onto Matrigel-coated Boyden chambers and treated with unsupplemented media or media containing 10 mM acetate, propionate, or butyrate. SW1116 invasion was quantitated by light microscopy and conditioned media were assayed by ELISA for MMP-1, 2, 3, and 9, TIMP-1 and 2, MMP/TIMP complex, and urokinase plasminogen activator (uPA). Although all 3 SCFA inhibited the invasion, butyrate was more potent than acetate or propionate. Butyrate inhibited the SW1116 invasion by 35±1% of control vs. 18±9% for acetate and 10±6% for propionate. MMP-2 was not modulated by any of the 3 SCFA, while MMP-1 was modulated only by butyrate and MMP-3 by propionate. Acetate did not modulate MMPs, TIMP-1, or uPA, but stimulated TIMP-2. Propionate and butyrate stimulated MMP-9 and TIMP-2 by 119-233% and both inhibited uPA by 8-16%. TIMP-1 was stimulated only by butyrate and inhibited by propionate. Only butyrate stimulated both TIMP-1 and TIMP-2. Thus, dietary fiber may protect against invasive colon cancer through stimulation of TIMP and inhibition of uPA activities, rather than through SCFA effects on the activities of the MMPs studied.

(c) 1998 Academic Press.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 15 MEDLINE on STN DUPLICATE 5
AN 97080953 MEDLINE
DN PubMed ID: 8922288
TI Progelatinase B forms from human neutrophils. complex formation of monomer/lipocalin with TIMP-1.
AU Kolkenbrock H; Hecker-Kia A; Orgel D; Kinawi A; Ulbrich N
CS Deutsches Rheumaforschungszentrum Berlin, Germany.

SO Biological chemistry, (1996 Jul-Aug) 377 (7-8) 529-33.
Journal code: 9700112. ISSN: 1431-6730.

CY GERMANY: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199702

ED Entered STN: 19970305
Last Updated on STN: 20000303
Entered Medline: 19970218

AB The three forms of neutrophil gelatinase B-monomer, homodimer and monomer/lipocalin complex-, were isolated from phorbol ester stimulated neutrophil granulocytes by chromatography on gelatin-Sepharose and heparin-Ultrogel. On average, about 50% of the monomer/lipocalin complex was found to be complexed with TIMP-1. After activation with trypsin monomer, homodimer and monomer/lipocalin complex displayed a specific activity of about 2000 mU/mg towards the substrate N-(2,4)-dinitrophenyl-Pro-Gln-Gly-Ile-Ala-Gly-Gln-D-Arg, whereas the monomer/lipocalin/TIMP-1 complex could be activated to a specific activity of only 200 mU/mg. The ternary monomer/lipocalin/TIMP-1 complex behaves like the progelatinase A-TIMP-2 complex and the progelatinase B-TIMP-1 complex in that it is an inhibitor for active metalloproteinases (MMPs) and, after activation, a gelatinase with a pronouncedly reduced activity. When the monomer/lipocalin/TIMP-1 complex **inhibits** an **MMP**, a quaternary complex monomer/lipocalin/TIMP-1/MMP is generated which after activation shows a sixfold higher proteolytic activity than the active ternary complex.